

and may explain the delayed bleeding in this patient.

This case emphasises the need for care in the selection of a site adjacent to the upper margin of the inferior rib when a pleural biopsy is performed.

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Thorax 1990;45:978-979

Chylothorax secondary to obstruction of the superior vena cava: a complication of the LeVein shunt

William H Warren, Jeffrey S Altman,
Stephanie A Gregory

Abstract

A case of thrombosis of the superior vena cava was complicated by bilateral chylothoraces and a widened mediastinum. Removal of a clotted LeVein shunt led to prompt resolution of the obstruction and chylothoraces.

Chylothorax is an uncommon result of obstruction of the superior vena cava and other veins, and reported causes include placement of central venous catheters.¹⁻⁶ We describe a case apparently caused by a peritoneovenous shunt.

Case report

A 43 year old man presented in 1984 with pancytopenia and splenomegaly. A diagnosis of hairy cell leukaemia was established by bone marrow biopsy. Splenectomy led to resolution of the pancytopenia. The patient developed malignant ascites and in June 1985 a LeVein peritoneovenous shunt was inserted. The ascites subsequently resolved. The patient was treated with interferon α_2 (3 million units subcutaneously three times a week) from July 1987 to September 1988; haematologically he remained stable and bone marrow biopsy showed evidence of complete remission.

The patient presented to us in September 1988 with a four week history of increasing

shortness of breath and a dry, non-productive cough, accompanied by swelling of the face, neck, and upper trunk. On examination the patient was afebrile and in no respiratory distress. He was noted to have oedema and plethora of the face and upper trunk and signs of a right pleural effusion. There was no abdominal tenderness or clinical evidence of ascites or organomegaly.

Investigations showed a haemoglobin level of 15.5 g/dl, a white cell count of $8.6 \times 10^9/l$ (normal differential count), platelets $440 \times 10^9/l$ and normal serum immunoglobulin concentrations with no monoclonal bands. The prothrombin time and partial thromboplastin time were normal; arterial blood gas analysis with the patient breathing room air showed that pH was 7.40, carbon dioxide tension 5.3 kPa and oxygen tension 10.3 kPa.

A chest radiograph confirmed the large right pleural effusion and showed a small left pleural effusion, a widened superior mediastinum, and the LeVein shunt at the junction of the superior vena cava and the right atrium. Computed tomography of the chest showed a filling defect in the superior vena cava around the shunt. There was no evidence of extrinsic compression of the superior vena cava, though mediastinal and axillary adenopathy was

Department of
Cardiovascular and
Thoracic Surgery
W H Warren

Department of
Hematology
S A Gregory

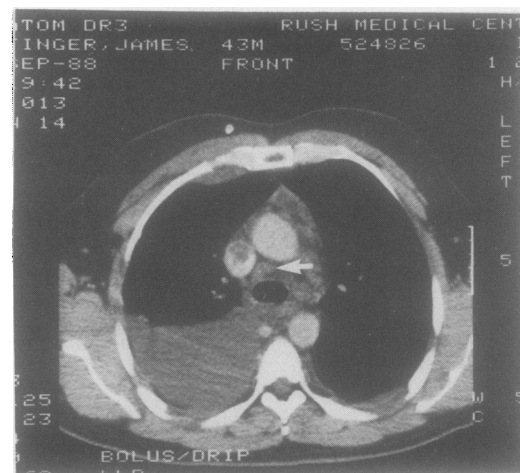
Rush-Presbyterian-
St Luke's Medical
Center

Rush Medical College
J S Altman (medical
student)

Chicago, Illinois, USA

Address for correspondence:
Dr W H Warren,
1725 W Harrison Street,
Chicago, Illinois 60612,
USA.

Accepted 20 June 1990



Computed tomogram showing bilateral chylothoraces, mediastinal lymphoedema, and lymphadenopathy (arrow). Note the thrombus in the lumen of the superior vena cava.

noted in addition to the bilateral pleural effusions (figure). Thoracocentesis established that the right pleural fluid was chylous (triglyceride 1925 mg/dl, cholesterol 78 mg/dl), white blood cell count $4.8 \times 10^9/l$ (4% neutrophils and 70% lymphocytes). Gram staining showed no organisms; the lymphocytes were polyclonal and there were no malignant cells. Tartrate resistant acid phosphatase lymphocyte staining was negative.

A bone marrow biopsy specimen was normocellular and showed no evidence of relapse of the hairy cell leukaemia. Ultrasound examination of the abdomen failed to detect ascites. The LeVeen shunt was removed under local anaesthesia and was found to be completely clotted. The patient was treated with heparin and warfarin. The diameter of his neck decreased, and his face and neck became less congested and plethoric. He was discharged in good condition but was maintained with anticoagulants for three months. Computed tomography performed nine months later showed complete resolution of the mediastinal widening and pleural effusions. A nuclear superior vena cavagram showed that the left subclavian vein and the superior vena cava had recanalised.

Discussion

Chylothorax has not been recognised as a manifestation of hairy cell leukaemia⁷; but T and B cell lymphomas may complicate treated hairy cell leukaemia and are a common cause of chylothorax. There was no evidence of a mediastinal mass in our patient, however, and the chylothorax and lymphadenopathy resolved promptly once the LeVeen shunt had been removed and the patency of the superior vena cava had been re-established.

Chylothorax has been identified as a complication of thrombosis of the superior vena cava in experimental studies¹ and by clinical observation.²⁻⁵ Blalock *et al*¹ in 1943 showed that acute interruption of the superior vena cava led to the development of a chylothorax in 60% of cats and dogs. Chylothorax has been reported in man as a complication of spontaneous thrombosis or obstruction of the superior vena cava, innominate vein, or subclavian vein.⁶ Other cases, occurring in newborn infants, children, and adults, have been attributed to the placement of central venous catheters.²⁻⁵

Peritoneovenous shunting is usually performed in patients with underlying liver disease and is more commonly associated with disseminated intravascular coagulation. When superior vena cava obstruction occurs it is often precipitated by thrombus ensheathing the venous arm of the shunt⁸; the resulting obstruction is usually limited to the superior vena cava, sparing the great veins into which the thoracic duct and collateral lymphatic channels drain. This may explain why lymphatic outflow obstruction is so uncommon, though obstruction of the superior vena cava has been reported in up to 25% of patients with long term shunts. To our knowledge,

this is the first case report of documented bilateral chylothoraces complicating thrombosis of the superior vena cava induced by a peritoneovenous shunt.

As lymph arising from the lungs is not chylous, it has been proposed that, in the setting of complete lymphatic outflow obstruction, chyle refluxes across incompetent lymphatic valves through the bronchomediastinal lymphangiectatic channels to the pleural space. In man this route has been documented by lymphangiography.^{9,10} Ligation of the superior vena cava in animals causes mediastinal tissues and lymph nodes to become considerably congested with chylous fluid.¹ The reversibly widened mediastinum and moderately enlarged lymph nodes in our patient were probably due to mediastinal lymphoedema.

According to previous reports, the syndrome of obstructed superior vena cava with chylothorax has a poor prognosis.^{3,4} Some have observed that, without relief of the venous obstruction, the lungs themselves become lymphangiectatic and this contributes to the long term morbidity.^{3,11} We believe that early relief of the superior vena cava obstruction in addition to dietary measures are important in the management of the chylothorax. Central venous catheters should be removed once they become non-functional. Systemic anticoagulation, streptokinase infusion, superior vena cava angioplasty or surgical removal of the thrombus (or some combination of these) have been performed to re-establish patency of the occluded superior vena cava and should be considered when this clinical syndrome occurs.^{8,12} Ligation of the thoracic duct at the diaphragmatic hiatus should be reserved for patients in whom re-establishing flow in the superior vena cava does not relieve the chylothoraces.

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